PROSPECTIVE STUDY ON IMPACT OF ULTRASOUND GUIDED FLANK DRAIN INSERTION ON THE PERIOPERATIVE OUTCOME OF CASES OF PERFORATIVE PERITONITIS WITH HEMODYNAMIC INSTABILITY

DISSERTATION SUBMITTED FOR

BRANCH I – M.S. (GENERAL SURGERY)

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THE TAMILNADU

DR.M.G.R.MEDICAL UNIVERSITY

CHENNAI

CERTIFICATE

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DECLARATION

I, **Dr.S.THALABATHIK KUMARA VIKRAM** hereby declare that, I carried out this work on "**PROSPECTIVE STUDY ON IMPACT OF ULTRASOUND GUIDED FLANK DRAIN INSERTION ON THE PERIOPERATIVE OUTCOME OF CASES OF PERFORATIVE PERITONITIS WITH HEMODYNAMIC INSTABILITY**" at the Department of General Surgery, Govt. Rajaji Hospital, Madurai, under the guidance of **PROF.DR.D.MARUTHUPANDIAN M.S.,FICS.,FAIS.,** Professor Of Surgery, during the period of August 2016 to July 2017.

I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other university or board either in India or abroad. This is submitted to the Tamilnadu DR. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulations for the M.S degree examination in general surgery (Branch I) to be held in May 2018.

Place: Madurai

Date: **(Dr. S.THALABATHIK KUMARA VIKRAM)**

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My patients, who form the most integral part of the work, were always kind and cooperative. I pray to God give them courage and strength to endure their illness, hope all of them go into complete remission.

And Last but not the least; I would like to thank My Parents, My Wife and THE ALMIGHTY for being with me throughout the study and supporting me without whom it would not have been possible.

CERTIFICATE – II

This is to certify that this disseratation work titled **PROSPECTIVE STUDY ON IMPACT OF ULTRASOUND GUIDED FLANK DRAIN INSERTION ON THE PERIOPERATIVE OUTCOME OF CASES OF PERFORATIVE PERITONITIS WITH HEMODYNAMIC INSTABILITY** of the candidate **Dr.S.THALABATHIK KUMARA VIKRAM** with Registration Number **221511121** for the award of **Master Degree** in Branch of **General Surgery**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **Two Percentage** of plagiarism in the dissertation.

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INTRODUCTION

INTRODUCTION

Hollow viscus perforation with secondary peritonitis is an abdominal emergency. Patients who are not fit for immediate surgery undergo resuscitation prior to laparotomy. In addition to iv fluids and inotropes, flank drain insertion under ultrasound guidance can effectively resuscitate these patients. Drainage of septic fluid under ultrasonic guidance decreases the sepsis, resulting in improvement of organ function. The procedure has been found to be quite beneficial as a supportive procedure in cases where immediate major surgical procedures like laparotomyare not possible due to comorbidities and unstable generalcondition. Further it can contribute to the postoperative outcome and have prognostic significance. In this study we compare the perioperative outcomes of patients undergoing/not-undergoing flank drain insertion during resuscitation prior to laparotomy.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

This study is concerned with the comparison of the perioperative outcomes of patients undergoing (test group)/not-undergoing (control group) flank drain insertion during resuscitation prior to laparotomy and the scope to implement the same.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Since the beginning of recorded medical history human beings have been confronted with the spectra of peritonitis. Accounts from a variety of early societies have little doubt that our ancestors recognized the value of the therapeutic drainage.

In a German translation of the writings of Hippocrates (400 B.C.), appears the first thorough description of a patient with peritonitis. "The patient looks sick and wasted, the nose is pointed, the temple sunken, the eyes lay deep are rimmed and dull, the face expresses fear, the tongue is furrowed, the skin shiny The patient avoids all movements and breathes shallow. The abdominal wall is rigid with muscular guarding and no bowel sounds can be heard. The pulse is quick and small. A hard tender mass in the hypochondrium is a bad prognostic sign if it involves the whole area. The presence of such a mass at the beginning of the fever indicates that death is imminent." This description is now a days called as "HIPPOCRATIC FACIES", and is still used for diagnosis of peritonitis.

The history of peptic ulcer dates back to 1500 B.C. when peptic ulcer and hemorrhage was noted from Egyptian Papyri. The history of

perforated peptic ulcers dates back to 1000 years ago, when people died of abdominal pain and vomiting and it was thought to be due to poisoning. It dates back to 1670, when king Charles 1 daughter, Henriette-Anne died suddenly after a day of abdominal pain and tenderness. Poisoning was suspected and an autopsy was performed, during which a small hole was found in the anterior abdominal wall, with signs of peritonitis.

The first description of gastric ulcer is attributed to physician Marcello Donati in 1586 arid first case of perforated gastric ulcer was recorded by Christopher Rrnylinson in England 1727.

In 1726, George Hamberger of Germany described a duodenal ulcer for the first time and in 1793, Jacopo Penada of Italy first recorded a duodenal ulcer perforation.

In 1843, Edward Crisp was the first to report 50 cases of PRU and accurately summarized the clinical aspects of perforation; concluding: "The symptoms are so typical, (hardly believe it possible that anyone can fail to make the correct diagnosis."

In 1881, Ludwig Rydygier, performed a successful resection of a pre-pyloric peptic ulcer.

In 1881, Theodor Biliroth, the father of surgical audit and father of abdominal surgery performed excision of distal part of stomach with an anastomosis of the gastric stump to the duodenum. In 1884, Dean performed closure of a duodenal ulcer and provided 'nutrition' by nutrient enemas and suppositories. In 1886, Reineke, did the first pyloroplasty.

Johan Mikulicz Radecki 6(1850 905), often referred to as the first surgeon who closed a perforated peptic ulcer (PPU) by simple closure said: "Every doctor, faced with a perforated duodenal ulcer of the stomach or intestine, must consider opening the abdomen, sewing up the hole, and averting a possible inflammation by careful cleansing of the abdominal cavity". In 1888, Mikulicz redefined the pyloroplasty done by Reineke.

In 1893, Barling of Britain treated perforated ulcer by closure and vigorous lavage of peritoneal cavity with large quantity of saline.

In 1893, Codivilla reportedly did the first gastrojejunostomy for a duodenal ulcer.

In 1896, Benette suggested sealing a large perforation with omentum.

In 1899, Kently performed gastric resection for a perforated peptic ulcer.

In 1929, Cellan Jones published an article entitled, "A rapid method of treatment in perforated duodenal ulcers". He suggested omentoplasty without primary dosing of the defect. His technique consisted of placing 4-6 sutures, selecting a long omental strand passing a fine suture through it, the tip of the strand is then anchored in the region of the perforation and finally the sutures are tied off.

It was not until 1937 that Graham published his results with a free omental graft. He placed three sutures with a piece of free omentum laid over these sutures, which are then tied. No attempt is made to actually close the perforation. The omental graft provides the stimulus for fibrin formation.

Jones- Graham technique has been the gold standard since then.

Wangenstein in 1935 first advocated non operative treatment for duodenal perforation.

In 1945, Taylor reported a senes of 28 consecutive cases of perforated peptic ulcers. Of those, 24 were treated non-operatively with intermittent gastric suction with three deaths.

In 1943, Dragsted and Owens introduced bilateral truncal vagatomy.

In 1948, Franksson of Stockholm first reported selective vagotomy. In 1965, Erik amdrup performed highly selective vagatomy.

Until the discovery of the role of H. pylori in gastric and peptic ulcers by Barry J Marshall and Robin Warren in 1982, stress and life style factors were believed to be the most important factor contributing to PUD and PPU.

In 1994 the National Institutes of Health Consensus Development Panel on Helicobacter pylon in PUD recommended that ulcer patients positive for H.Pylori should be treated with antimicrobial agents.

In 1992, Feliciano' described 5 decisions facing the surgeon in the situation of perforated peptic ulcer. Those decisions are as follows:

1. Is the performance of an operation indicated?
2. Is an omental plication sufficient or is a definitive ulcer operation indicated?
3. Is the patient stable enough to undergo a definitive ulcer operation?
4. Which definitive ulcer operation is indicated?
5. Should the availability of newer medical options influence the choice of operation?

1985-Johansson.B, Gilse.H. Described a laparoscopic technique for closure of perforated peptic ulcer.

Since the 90's, there has been various reports of laparoscopic closure of a perforated peptic ulcer. Laparoscopic surgery offers several advantages.

First of all a laparoscopic procedure serves as a minimal invasive diagnostic tool.

A meta-analysis by Lau H, published in 2004, reported benefits from laparoscopic repair as postoperative pain reduction and less consumption of analgesics and a reduction in hospital stay. Also a reduction in wound infections, burst abdomen and incisional hernia due to shorter scars has been noted. Avoiding upper laparotomy might lower the incidence of postoperative ileus and chest infections.

Drawbacks are a prolonged operating time, higher incidence of re-operations due to leakage at the repair site and a higher incidence of intra-abdominal collection second to inadequate lavage. If the presence of these fluid collections have any clinical relevance is unclear. The higher incidence of leakage might be caused by the difficulty of the laparoscopic

Suturing procedure. This emphasises the need for a dedicated laparoscopically trained surgeon to perform this procedure.

Alternative techniques to simplify the suturing process have been thought of. Some laparoscopic surgeons use omentopexy alone. Some surgeons have sutured the falciform ligament over the perforated ulcer. Sutureless techniques of laparoscopic repair of perforated duodenal ulcers eliminate the need for laparoscopic suturing and have been shown to decrease operative time. These include the use of a piece of gelatin sponge shaped into a cone and placed into the ulcer with prewarmed fibrin sealant injected around the plug described by Tate JJ et al. The use of fibrin glue alone to seal the hole with omentum, was described by Mouret P in 1990. The downside of this technique is that it only can be used to close small perforations. To overcome this problem a biodegradable patch made of Lactide Glycolide-Caprolacton that can be cut into any desirable size, has been described by Barlette Met.al.

Gasless laparoscopic closure of duodenal ulcer perforation was reported in 1997 by Viani et.al. This provides a clear field of vision in the abdomen equal to that created by the traditional C02 technique. Because there is no pneumoperitoneum to maintain, the gasless technique permits a constant irrigation and suction of the

abdominal cavity, a wide peritoneal lavage, and the continuous suction of fluid, blood, smoke, and humidity.

In 1996, Halkic.N. Pescatore. P. and Gilleton described a new method combining laparoscopy and endoluminal endoscopy, designed to ensure complete closure of the perforation. Laparoscopy and endoluminal endoscopy are used concomitantly for closure of the perforation by an omental plug attracted into the digestive tract.

A multi-centre randomised control trial, the LAMA trial 18, comparing open correction of perforated peptic ulcer with laparoscopic repair, included 101 patients from 9 medical centres, during 1999 to 2005. 52 were treated by laparoscopic surgery and 49 patients received an upper midline laparotomy. Results showed that the laparoscopic procedure takes longer surgery time, but patients in this group had less postoperative pain. Concluding that laparoscopic closure of perforated peptic ulcer was a safe procedure.

In a retrospective cohort study on outcome of Laparoscopic simple closure of perforated duodenal ulcer, by Hisham A 19 et.al, 191 patients were studied and the method was found to be safe and reliable with low morbidity.

ANATOMY AND RELEVANT PHYSIOLOGY OF DUODENUM

The word Duodenum is a Latin corruption of the great word "dedekadaktulas" meaning 12 fingers, indicating it's length,12 finger breadths.

DEVELOPMENT OF DUODENUM

Early in the 4th week, the duodenum begins to develop from the caudal or distal part of the foregut, the cranial or proximal part of the midgut. The junction of the two parts of the duodenum is just distal to the origin of the bile duct. The developing duodenum grows rapidly, forming a C-shaped loop that projects ventrally. As the stomach rotates, the duodenal loop rotates to the right and comes to lie retroperitoneally.

Duodenum is a C-shaped, first, shortest [25cms) and most fixed part of the small intestine. It extends from the pylorus to the duodeno jejunal junction, making C-shaped curve which is occupied by the head of the pancreas and lies entirely above the level of the umbilicus.

Duodenum is divided into four parts

FIRST I SUPERIOR PART: 5cms long, it begins at the pylorus, runs upwards and backwards and lies on the trans-pyloric

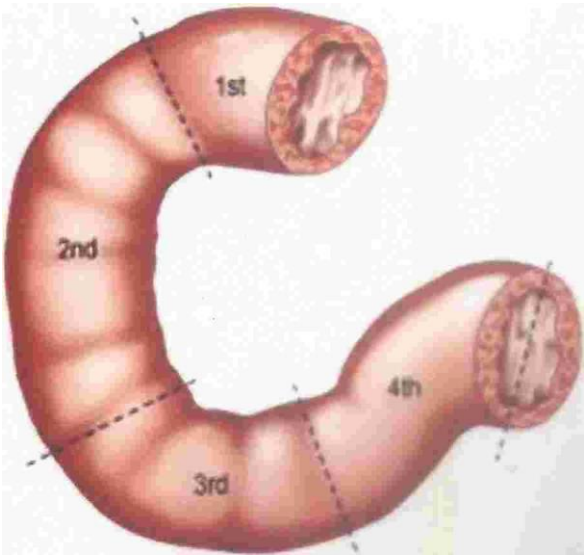
plane. The 1 inch is covered with peritoneum on the front and back and can be moved with the stomach.

SECOND / DESCENDING PART: 8cms long. Forms the C loop. About half way down on its medial border, the bile duct and the pancreatic duct open into the major duodenal papilla.

THIRD / HORIZONTAL PART: It is 3 inches I 8cms long, runs horizontally and to the left on the subcostal plane and is crossed by the root of the mesentery.

FOURTH / ASCENDING PART: 5cms long, shortest part of the duodenum, ends at the duodenojejunal flexure which is held in position by ligament of Treitz, attached to the right crus of the diaphragm.

FIG 1: PARTS OF DUODENUM



BLOOD SUPPLY:

The duodenum is supplied by the superior and inferior pancreaticoduodenal arteries. The first 2 cm receives small branches from common hepatic, gastroduodenal, superior pancreaticoduodenal, right gastric and right gastroepiploic arteries.

Duodenum drains into superior and inferior pancreaticoduodenal vein

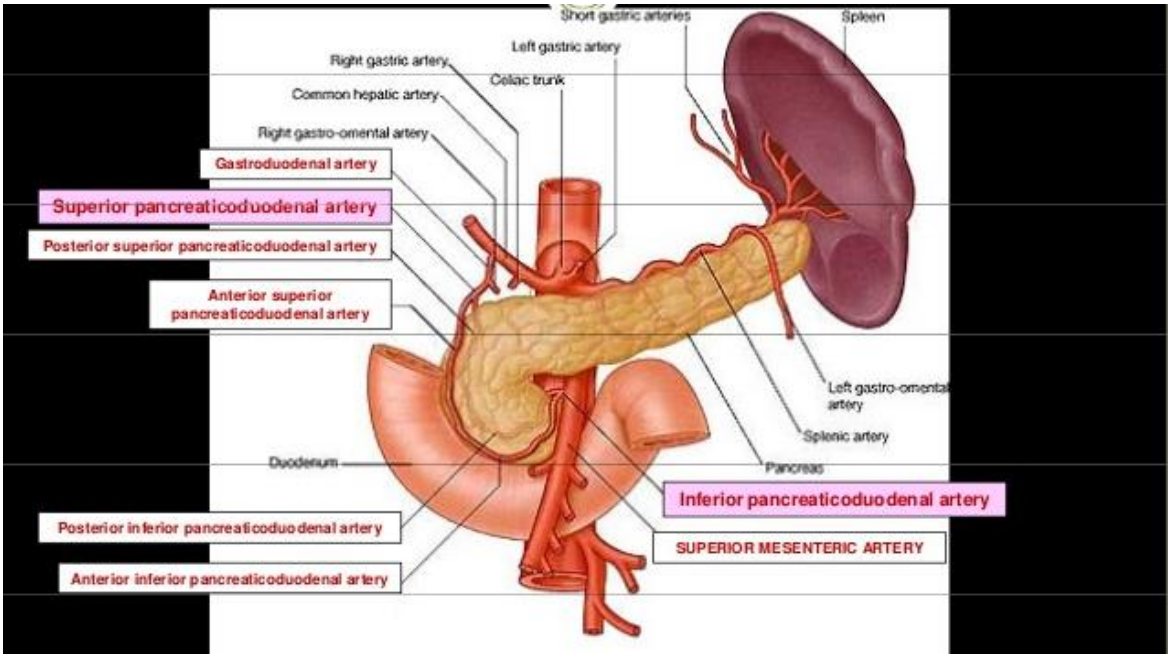


FIG 2: BLOOD SUPPLY OF DUODENUM

LYMPHATIC DRAINAGE:

Duodenal lymphatics run to anterior and posterior pancreatic nodes which drain into the supra-pyloric, infra-pyloric, hepatoduodenal, common hepatic and superior mesenteric nodes.

INNERVATION:

Sympathetic and parasympathetic fibers from the celiac and the superior mesenteric plexus.

PHYSIOLOGY OF DUODENUM

The functions of the duodenum can be studied under 3 headings:

Secretory function:

The secretion is alkaline- pH 8 to 8 .2 and contain bicarbonate, contains amylase and enterokinase. These enzymes originate in the columnar cells of the duodenal epithelium and are not a product of Brunners glands.

Endocrine secretions of duodenum:

- Secretin
- Cholecystokinin-pancreozymin
- Enteroglucagon
- Enterogastrone

The presence of acid chyme m the duodenum stimulates the secretion of above enzymes.

Motility of duodenum:

There are two principal types of movements:

- Rhythmic segmentation- not well developed in duodenum.
- Peristalsis-about tenfold faster than the rest of the small intestine, at a rate of 17-18 per minute.

Absorption in duodenum:

The following substances are absorbed in the duodenum

1. Glucose
2. Iron
3. 3. Water soluble vitamins -Folic acid, Riboflavin, Pyridoxine and Ascorbic acid

DUODENAL ULCER

Acid peptic disorders are group of ulcerative disorders of the gastrointestinal tract involving principally the most proximal part of duodenum, stomach, lower end of esophagus and jejunum after surgical anastomosis to stomach or rarely the ileum adjacent to the Meckel's diverticulum due to ectopic gastric epithelium.

Approximately 98% - 99% of the peptic ulcers occur in the 1 part of duodenum or in stomach. About 5% of individuals with gastric ulcer develop duodenal ulcer, but 20% of those with duodenal ulcers develop gastric ulcers.

The pyloric channel which is 1-2cms in length is the narrowest portion of the gastric outlet, because of their gastric acid secretary characteristics and clinical features pyloric channel ulcers are grouped with duodenal ulcers rather than gastric ulcers. Ulcers in this location often produce symptoms similar to duodenal ulcers.

EPIDEMIOLOGY:

Annual incidence is about 1.8% or roughly 500,000 thousand new cases per year with about 4 million ulcer recurrences per year. It is more prevalent in the developing countries and in low socioeconomic class. Incidence is higher in patients with smoking, alcoholism, chronic NSAIDs users and in persons with type A personality. Genetic influence plays some role in the peptic ulcer pathogenesis, is clearer in cases of duodenal ulcers. Duodenal ulcers are 3 times more common in 1 degree relatives of ulcer patients than in general population) a 50% concurrence of duodenal ulcers in monozygotic twins but only 14% in dizygotic twins is noted. An increased

incidence of HLA-65 antigen has also been identify in white males with duodenal ulcers. Individuals with blood group O are about 30% more likely to develop duodenal ulcer than those with other blood groups.

Increased use of NSAIDs, corticosteroids, spicy & smoked foods, alcohol consumption and smoking are also important in pathogenesis of duodenal ulcers. Duodenal ulcer is also more common in patients with alcoholic cirrhosis, chronic renal failure and chronic obstructive pulmonary diseases and in patients with hyperparathyroidism.

ROLE OF HELICOBACTER PYLORI:

The phrase 'NO ACID - NO ULCER" does not hold well now a days because peptic ulcer is now considered more as an infective disease caused by H.pylori. In 1983 Warren and Marshall first reported isolation of H. Pylon from the mucosal biopsy of patients with peptic ulcer diseases.

H. pylori is a small spirally curved gram negative microaerophilic rod with multiple polar flagellac. Infection of a population increases with age and is inversely related to socioeconomic status of the population.

This organism is the major cause of peptic ulcer disease not associated with the use of NSAIDs, humans are major reservoirs of this organism which colonizes in the stomach much frequently in the antrum, route of transmission is feco-oral and oro-oral routes.

PATHOGENESIS OF PEPTIC ULCER DISEASES

All peptic ulceration probably arises because of an imbalance between the aggressive action of acid and pepsin secretion and the normal defenses of the gastro duodenal mucosa. For duodenal ulcer major causal influence appears to be exposure of the duodenal mucosa to the excess amount of acid and pepsin. For gastric ulcer the major causal influence appears to be break down in the gastric mucosal defenses against acid and pepsin. The hyper secretion is related to an abnormally large total mass of parietal cells in the gastric mucosa due to either increased responsiveness of parietal cells to secretory stimuli or lack of normal regulatory controls. Increased sensitivity of the parietal cells to gastrin stimulation may also be involved. Individuals with total achlorhydria never develop a duodenal ulcer.

Defects in the defense mechanism such as deficiencies in mucosal cell renewal, mucous production, elaboration of bicarbonates

and production of prostaglandins, play major role in peptic ulcer disease pathology.

Pathogenesis of duodenal ulcer due to H. pylori ranges from asymptomatic gastritis to peptic ulceration and gastric carcinoma.

H.pylori colonizes in the gastric epithelium causing type-B gastritis by which it reduces the resistance of gastric mucosa to attack by acid and pepsin, resulting in gastric ulcer. Although it normally resides in the stomach it leads to causation of duodenal ulcers, which can be explained by the fact that antral infection impairs the inhibitory feedback of acid secretion thus promoting duodenal ulcerogenesis by increasing duodenal acid load.

- By increasing the acid secretion: H. pylori produces urease which hydrolyses urea, resulting m production of ammonia, a strong alkali, which causes release of gastrin from antral G-cells causing hypergastrinemia leading to gastric acid hyper secretion.

- By disrupting gastric mucosal barrier

- By secretion of various enzymes: such as urease, catalase, lipase, phospholipase, porms, proteases, hemolysins and

alkaline phosphatase leading to impaired acid-pepsin balance and local tissue injury.

- By inducing inflammation in gastric epithelium [Wyatt & Dixon hypothesis]

H. pylori infection has also been implicated as a risk factor in gastric carcinoma and low grade gastric lymphoma. Now WHO has described H. pylori as class-I carcinogen.

COURSE OF THE DISEASE: Irrespective of treatment peptic ulcer takes one of the following courses during the period of its progression:

- Healing,
- Chronicity and complications:
    1. Hemorrhage,
    2. Perforation,
    3. Cicatricial contraction and Carcinomatous changes.

PATHOPHYSIOLOGY OF PERFORATION OF DUODENAL ULCER:

Perforation is the natural termination of an ulcer which continues to penetrate deeper tissues. Recently, it was found that duodenal ulcer perforations greatly outnumber gastric ulcer perforations.

Incidence of perforation is approximately 7 -10 cases per 1 lakh population per year. Perforation occurs in 10%-15% of established cases of peptic ulcers and is the first manifestation in 2% of patients.

Anterior ulcers tend to perforate because of the absence of protective viscera, in contrast to the bleeding ulcers that are usually situated posteriorly in <10% of patients with high death rate.

Boyd was of the opinion that perforation is more common in ulcers of short duration, from few days to few weeks, in which there is rapid penetration of deeper tissues. Ulcers of long duration with abundant scar tissue are less likely to penetrate which is prerequisite for perforation. Ulcers with continuous symptoms are more harmful than ulcers with history of remissions.

In a study of 201 patients with perforation by John Gelmon in 1953, 119(58%) were found to have acute and 82(42%) had chronic duodenal ulcers.

In a study conducted by Illingworth in 197 5 about 90% of cases perforation has resulted from sloughing off of the floor of the ulcer.

In Lawden's study where he performed primary gastrectomy and on subjecting them for HPE he concluded that out of 41 cases, 22 were undoubtedly chronic, 16 were grouped as subacute and remaining 3 as acute.

PRE DISPOSING FACTORS FOR PERFORATION

AGE: Common in both younger patients and in elderly patients, no age is exempted, but perforation is rare in childhood.

A large study conducted in Great Britain by J. Higham and colleagues from 1989 to 1999 showed that perforation was more common in patients aged >65yrs.

SEX: Perforation is more in men compared to women.

Mckay and Mckay in their study gave a male: female ratio of 4.1: 1.

OCCUPATIONAL INCIDENCE: Perforations are more common m those who were engaged in heavy manual work.

In 1960 Kozali and Mayer reported population incidence m 1904 perforations which is as follows,

- Unskilled-27.9%
- Semiskilled-14.5%
- Skilled-12.9%
- Dependents- I 1. 0%

DIURNAL VARIATION: Jamieson in 1944, Strang and Spencer in 1950, found increased incidence of perforation in the afternoon and evening and less incidence during nights.

RELATION TO MEALS: Jamieson in 1944, Been in 1943, stated that perforation is more common 2-3 hrs. after a meal, which could be due to over distension of stomach. Dr. S. S. }-Hussain in 1965 reported that perforation is more common immediately after food.

H.PYLORI AND PERFORATION: H. pylori eradication dramatically reduces recurrence of ulcer and related complications such as hemorrhage and perforation.

MEDICAL TREATMENT: Abrupt cessation of H2 receptor blockers precipitated perforation in previously diagnosed peptic ulcer patients in a study done by Wallace in 1977, which is attributed to acid rebound.

PREGNENCY AND PERFORATION: In a study conducted by S Way in 1945, he noted that incidence of peptic perforations were high during pregnancy which is attributed to change in hormonal milieu in that period.

RELATION TO PHYSICAL STRESS: Debakey in 1940 reported that trauma in around 4% of patients played a role in perforation of peptic ulcers, whereas Jamieson in 1944 in his study concluded that severe exertion plays little role in perforation of peptic ulcer diseases.

OTHER PREDISPOSING FACTORS: Upper respiratory tract infection, fatigue, exposure to cold damp weather, worry and anxiety, alcoholism, heavy smoking and failure to maintain diet control are some of the contributing factors during the period of exacerbation.

PATHOLOGICAL COURSE

At the onset of perforation there is sudden spillage of the duodenal and gastric contents into the general peritoneal cavity and it results in chemical peritonitis. The degree of involvement of the peritoneal cavity by bacteria is always uncertain. It is suggested that at first the visceral contents are sterile and the infective peritonitis in the early case is unlikely. However it depends on the general condition of the patient and his resistance to infection.

Perforation of peptic ulcer may be classified as,

- Acute perforation,
- Sub acute perforation,
- Chronic perforation
- Perforation associated with haemorrhage
- Pseudoperforation and rarely
- Perforation of an intrathoracic gastric ulcer

ACUTE PERFORATION: The ulcer perforates and the general peritoneal cavity becomes flooded with gastric and duodenal contents, causing chemical peritonitis.

The clinical features vary according to the stage of perforation, the course is divided in to 3 stages" of variable duration:

I. Primary Stage: (Irritation of the peritoneum) - The stage of peritonism. It occurs due to the sudden leak of fluid into the peritoneal cavity. The pain may be in the right hypochondrium and epigastrium. The pain may radiate to the back in case of a perforation into the lesser sac. There may be shoulder tip pain with the pain, later becoming generalized. There may be a syncopal attack. There may be associated nausea and vomiting. tachycardia. Abdominal guarding and rigidity may be evident, but may be absent in cases of pelvic peritonitis. Liver dullness obliteration is based on the amount of gaseous escape. Abdomen may be lax in multipara and elderly.

A leaking duodenal ulcer can give rise to drainage along the right paracolic gutter "Moynihan's Gutter Sign" or 'Vincent's Appendicitis".

2. Secondary Stage (Stage of delusion or reaction): It last for six hours. The escaping fluid is neutralized by peritoneal reaction and gives the doctor a sense of false security as the signs and symptoms abate. Liver dullness may be obliterated, muscles may be soft. Shifting dullness and paralytic ileus set in. Per abdomen examination elicits tenderness and chest x-ray shows pneumoperitoneum in 70% of cases.

3. Stage of Bacterial Peritonitis: The leaking fluid carnes with its bacteria from the oesophagus, stomach, small and large intestines and transudation ensues. The fluid becomes purulent and the bowels oedematous. Patient has fever with chills, hypoxia and renal failure passing into a stage of paralytic ileus if untreated. Patient is in septic shock with paralytic ileus, He has a characteristic appearance, the 'facies hippocratica' with an anxious look, wide eyes, rising pulse rate and falling blood pressure.

SUB ACUTE PERFORATION: An ulcer may perforate and the perforation may rapidly seal off before there is spillage of gastric and duodenal contents into the peritoneal cavity. There is sudden onset of acute abdominal pain often more severe at the right upper quadrant. On examination, there is local tenderness and rigidity in the epigastrium or right hypochondrium, but rest of the abdomen is soft to palpate and non• tender. After an hour or two, with bed rest pain usually subsides.

CHRONIC PERFORATION: When an ulcer perforates into the area that is walled off by adhesions or by adjacent viscera such as liver, colon or greater omentum or into the omental sac, a chronic abscess may develop, giving rise to considerable confusion in diagnosis. May present as sub-phrenic abscess. USG of

abdomen is the most reliable investigation in diagnosing intraperitoneal abscess.

PERFORATION ASSOCIATED WITH HAEMORRHAGE: Perforation in association with massive hemorrhage is grave but fortunately its incidence is rare.

RARE TYPE OF PERFORATED PEPTIC ULCERS: A peptic ulcer in a Meckels diverticulum, or an intestinal duplication which perforates. Multiple simultaneous perforations occur in less than one percent of all cases.

CLINICAL FEATURES OF PEPTIC ULCER PERFORATION

AGE: Peptic ulcer perforation is rare before adolescence, common in 30 - 40 yrs. age group.

SEX: More common in men than women.

HISTORY OF PRESENTING ILLNESS:

- Time of onset: Very often the patient is able to exact the time of onset of symptoms which is characteristic of perforation. Perforation is common particularly after an exertion in the evening.
- Mode of onset: Sudden in onset.

- Type of Pain: Pain is tearing type in the abdomen, intense in the right hypochondrium and epigastrium which then spreads to the whole abdomen.
- Shifting of pain: Pain shifts to right iliac fossa as the fluid flows down along right paracolic gutter to settle in the right iliac fossa, thus mimicking appendicitis.
- Radiation of pain: Pain in peptic ulcer perforation radiates to tip of the shoulder.
- Nausea: may be present in some patients.
- Vomiting: Initially, reflex vomiting occurs due to irritation of nerves in the peritoneum and the mesentery. In the later stages, vomiting is due to toxin action at the medullary center, causing paralytic ileus. The vomiting then contains undigested food particles and occasionally blood, when hemorrhage is present.
- Bowel movements: In the later stage, there may be desire to defecate, due to irritation of rectovesical pouch by irritant fluid. Melena occurs when hemorrhage is associated with the perforation.
- Urine output: Oliguria, if the patient is in shock.

PAST HISTORY: In 80% of patients, there is a history of dyspepsia of variable duration and in about 59%, the perforation is recurrent. In the rest of the cases, the perforation may be the first clinical manifestation of a silent peptic ulcer.

PHYSICAL EXAMINATION:

- General appearance: in the initial stages of perforation, patients face is pale and livid with sweating.
- Decubitus: The patient lies supine, is rigid and immovable and refuses of any attempt to shift his posture.
- Pulse: Initially normal, becomes rapid as peritonitis sets in and thready as the prognosis worsens.
- • Respiration: No change initially, becomes rapid and shallow as peritonitis sets in or in perforation with hemorrhage.
- Temperature: Initially normal, rises with the onset of peritonitis.
- Tongue: Becomes dry and brown as peritonitis sets in.

EXAMINATION OF THE ABDOMEN:

- Respiratory movements: thoracic movements predominate over the abdominal movements during respiration.
- Palpation of the abdomen: rigidity of the abdomen is constant, continuous and board like. It is due to reflex

contraction of abdominal muscles with predominance in the epigastrium and right hypochondrium. Rigidity is less in poor risk patients.

- Liver dullness: obliteration of liver dullness elicited in the front and in the midaxillary line, is characteristic of perforation in the second stage.
- Free fluid: present in variable degree in many acute abdominal conditions. When internal hemorrhage is excluded, fluid of appreciable amount points out the provisional diagnosis of perforation in acute abdomen.
- Digital rectal examination: There may be fullness in rectovesical or rectovaginal pouch.

INVESTIGATIONS

The diagnosis is easy, but difficulties are experienced at times when other conditions mimicking perforation are met with.

Erect X-ray abdomen:

In plain X-ray erect abdomen in about 80% of cases, free air under both domes of diaphragm is evident, According to Krupasindu Panda and Chakraborty's study conducted in 1976, 85% of patients demonstrated air under diaphragm. According to Peter et al in 1955

and Isle in 1951, X-ray should be taken only after complete aspiration of food material from the stomach, as the mucous or food material may block the leak, hence results in absence of sub diaphragmatic gas shadow.

Gastroduodenogram:

Some clinics have used X-ray pictures of abdomen following injection of 60ml of 50% gastrograffin through nasogastric tube. The dye escapes through perforation, thus enabling to demonstrate the site and size of the perforation, evidence of chronicity, associated gastric ulcer and if any second ulcer present.

Ultrasound examination:

Ultrasonography of abdomen is performed using a convex multi-frequency probe (3.5 -5 MHz) Evidence of intraperitoneal free fluid with air pockets arid internal echoes with reduced intestinal peristalsis gives an indirect evidence of perforation.

Computed Tomographic Scanning:

Because of classical presentation in most patients, CT- Scanning is rarely required for diagnosis. However, patients with perforated duodenal ulcer who are on steroid therapy or who are hospitalized for other abnormalities may develop occult causes of abdominal pain and

sepsis, and such patients may be diagnosed by Gastrograffin swallow and CT• Scannin9 to determine the cause of occult abdominal sepsis.

Serum amylase:

It is usually normal, but may be raised to a small extent. Normal value of serum amylase is 80 -180 somogyi units. Above 200 units is considered pathological. Mortality is high for gastric and duodenal perforation with high serum amylase.

Helicobacter Pylori infection diagnosis

The diagnosis of this infection is done by following methods:

Noninvasive methods:

- Serology ELISA.
- Urea breath test Invasive methods:
- Rapid urease test
- Histopathological examination
- Culture.

Abdominal paracentesis:

Diagnostic peritoneal tap is a simple procedure, which can be done quickly in case of suspicious hollow viscus perforation. Four quadrant abdominal paracentesis has to be done.

DIFFERENTIAL DIAGNOSIS:

An early presentation with typical symptoms and signs usually within 12 hours of perforation causes minimal problems with diagnosis.

The differentials to be considered after a late presentation are as follows,

Conditions requiring operation:

Acute Appendicitis: Pain starts in the umbilical region and radiates to the right iliac fossa and is associated with vomiting and fever Mc Burney's point tenderness, Pembertons' sign; Cope's psoas tests are usually positive. A perforated appendix may cause problems with diagnosis.

Intestinal Obstruction:

Based on the level of obstruction, it presents with pain; abdominal distension, vomiting, constipation and obstipation. Hernias are the most common cause of intestinal obstruction if post - operative adhesions are not considered. Obstructed bowels can also perforate.

- Ruptured ectopic pregnancy: Associated with pallor, collapse, shock, history of amenorrhoea, vaginal spotting and bogginess in the fornices.

Conditions where the progress can be watched and operated upon if required:

- Acute cholecystitis: Usually occurs in females over 40 years and they are obese. May be associated with jaundice and biliary colic. Murphy's sign and Charcot' s pentad may be present. Ultrasonography is diagnostic.
- Acute pancreatitis: May be in an alcoholic or associated with gallstones. Back pain, vomiting and epigastric tenderness are prominent.
- Primary Peritonitis: Usually presents in an ascetic patient or asymptomatic patient with fever, pain and vomiting.

Conditions to be managed conservatively:

Medical:

Basal pneumonia,

Myocardial infarction

Diaphragmatic pleurisy

Acute intermittent porphyria

Diabetic ketoacidosis

Renal failure Radiculopathy

Fracture of the spine

Surgical

Mesenteric lymphadenitis

Inflammatory bowel disease.

TREATMENT

The perforation of pyloro-duodenal ulcer and gastric ulcer is a common problem The most important and immediate step in the management is adequate resuscitation of the patient on admission.

The following operative procedures have been described for the treatment of perforated duodenal ulcer:

1. Simple closure of perforation, together with technical modifications such as the use of an omental patch (Graham's patch), has been the mainstay of surgical treatment of perforation of duodenal ulcer in most centres.
2. Definitive ulcer operations for perforated duodenal ulcer
    - Simple closure of perforation with drainage procedure like gastroenterostomy with or without vagotomy
    - Simple closure of perforation with pyloroplasty and vagotomy
    - Gastric resection with or without vagotomy

3. Laparoscopic closure of perforation

IMMEDIATE MANAGEMENT (RESUSCITATION):

- If patient is in shock, elevate foot end of the bed, start intravenous fluids immediately with Dextran or Saline/Plasma expanders. If the patient's blood pressure is not adequately picking up,inotropic support can be given.additionallyb/l flank drain insertion can be done if possible under ultrasound guidance
- A nasogastric tube is passed and the contents of stomach aspirated and repeated every half an hour to prevent:
    - further soiling of peritoneum
    - aspiration of gastric contents into lungs
    - And to decompress the stomach.
- Bladder catheterization done in all patients to monitor urinary output.
- Blood sample collected for grouping and cross matching, complete haemogram, blood urea, serum creatinine and serum electrolyte study.
- Blood pressure and pulse rate and urinary output should be recorded at half hourly intervals.

- Appropriate antibiotics (broad spectrum) should be given. Third generation cephalosporins and metronidazole are preferred.
- Preparation of abdomen to be done.

PROCEDURE FOR FLANK DRAIN INSERTION:

- Sterile aseptic precautions taken.
- Patient painted and draped.
- The skin, subcutaneous tissue are infiltrated with 1% injection lignocaine.
- Ultrasound guidance obtained.
- A short skin incision is made with a no. 1 knife at the flank
- Using the long curved artery forceps the muscles are separated and reached up to the peritoneum. The chest tube held by an hemostatic forceps is thrushed through the peritoneum puncturing it and the tube is inserted into the peritoneal cavity.
- The tube is clamped with a hemostatic forceps and the closed end is cut off and the tube is connected to a bag.

OPERATIVE MANAGEMENT SIMPLE CLOSURE:

After adequate resuscitation patient can be posted for surgery.

Anaesthesia: General anaesthesia by IV thiopentone and scolene, endotracheal intubation and maintenance by N2 and 02.

Procedure: With the patient in supine position, abdomen is opened in layers with upper midline incision. Bailey points out that in 10% of cases a muffled pop of escaping gas can be heard on opening the peritoneum. Free fluid is sucked and peritoneum mopped with moist packs. The stomach is held near the greater curvature and perforation searched. It will be found in the first part of the duodenum anterior surface usually or it may be in the distal stomach. Perforation found is sutured with interrupted sutures using silk or vicryl round bodied, along the long axis of the bowel. After through peritoneal toilet, 2 abdominal drains are placed, one into the Morrison's pouch and the other in the pelvis and the incision closed in layers.

METHODS OF CLOSURE OF PERFORATION

1. Simple closure:
    a. Indicated in small ulcers with little induration and healthy tissue around.
    b. Converts ulcer into a linear scar.
    c. More rapid healing.

d. Early remission of symptoms.
e. Delayed absorbable /non absorbable suture materials can be used.
f. Purse string sutures should be avoided. g. Inversion I eversion should be avoided.
g. Sutures should be applied in the long axis of the bowel to avoid narrowing of the lumen.
h. Omentum is used for the reinforcement of the perforation.

2. Cellan Jones technique (1929) and Graham's technique (1937): Here greater omentum is placed over the perforation and perforation is sealed. In Jones technique edge of perforation is sutured with the free omental flap where as in Graham's technique greater omentum is placed over the perforation and the sutures taken along the long axis of the bowel are enforced by placing the omentum over the perforation.

3. Dragging the omentum into the perforation and plugging into the Ryles tube.

4. Use of rectus muscle to seat the perforation.

DEFINITIVE SURGERY:

This was advocated as it was found that patients treated with simple closure have a severe relapse of the disease in >50% of cases in 5 years follow-up.

INDICATIONS OF DEFINITIVE SURGERY:

Absolute indications:

- Coexistent of perforation and haemorrhage.
- Previous operation for perforated duodenal ulcer.
- Perforated gastric ulcer with suspicion of malignancy.
- Coexistence of haemorrhage and perforation.
- Perforation of ulcer during medical treatment.
- Combined gastric and duodenal ulcer, one of which has perforated.

Relative indications:

- Young patient less than 45 yrs.
- Smoker.
- Absence of purulent peritonitis.
- If patient has reported within B hrs of perforation.
- Minimal peritoneal soiling.

CONTRAINDICATIONS FOR DEFINITIVE SURGERY:

- More than 24 hrs. of perforation.
- Poor risk patient.
- Concurrent medical illness.

ADVANTAGES OF DEFINITIVE SURGERY:

- Re-perforation is avoided.
- Second operation is avoided.
- Gastric stasis after simple closure is avoided.
- Post-operative pyloric stenosis and obstruction due to inflammation and edema are avoided.
- In hemorrhage with ulcer perforation hemorrhage is cured.

DISADVANTAGES OF DEFINITIVE SURGERY:

- More operative trauma to the patient.
- May be unnecessary in 10 - 15% of patients.

TYPES OF DEFINITIVE SURGERY:

- Truncal vagotomy with gastrojejunostomy.
- Antrectomy with vagotomy.
- Closure with highly selective vagotomy.
- Partial gastrectomy with vagotomy.

- Pyloroplasty with vagotomy.

LAPAROSCOPIC CLOSURE OF PERFORATION

RECENT DEVELOPMENTS in minimal invasive surgery now allows laparoscopic approach to the patient with perforated duodenal ulcer.

In patients presenting within 24hrs of onset of symptoms, without any comorbid conditions and with minimal contamination, laparoscopic perforation closure is preferred modality now a days. Requirement of analgesics, hospital stay, and economic burden will be reduced greatly along with best cosmetic results.

The perforation can be approached usmg 4 ports (1 is 1 Omm umblical port for camera, another 1 Omm working epigastric port and remaining 2 are of 5mm which are functional ports), additional ports can be used if required.

The perforation can be closed by any of the following methods:

- Fibrin glue for minute perforation,
- Simple closure with Graham's omental patch and copious irrigation of the abdominal cavity (this procedure was followed in our study),

- Automatic staples can be applied via laparoscopic device (BJS, Dec 1993).
- A proximal gastric vagotomy or Taylor procedure (anterior seromyotomy and Truncal vagotomy) may be performed.

POST OPERATIVE MANAGEMENT

- NIL BY MOUTH till the intestinal sounds regained.
- Nasogastric tube aspiration till it becomes less than 150 ml I day and bowel sounds recover.
- Antibiotic covering anaerobic and aerobic organisms.
- Intravenous fluid therapy.
- Proton pump inhibitors.
- Chest care.
- Drainage tube care.
- Watch for any evidence of intra-abdominal collection m case of postoperative fever.

POST OPERATIVE COMPLICATIONS:

- Pulmonary complications like atelectasis, pneumonia.
- Residual abscesses like sub phrenic abscess, pelvic abscess.
- Peritonitis.

- Paralytic ileus.
- Early reperforation and leak, duodenal fistula.
- Deep vein thrombosis and pulmonary embolism.
- Renal failure.
- Mediastinitis.

MANAGEMENT OF EARLY REPERFORATION AND LEAK:

- Priority towards fluid and electrolyte management.
- Nasogastric aspiration.
- Proton pump inhibitors.
- Antibiotics.
- Nutrition of the patient, ideally total parenteral nutrition (TPN) or feeding jejunostomy.

CAUSES OF LEAK:

- Old age patients.
- Large perforation.
- Inadequate closure.
- Difficulty closure with friable margin.
- Late presentation to hospital after perforation.

Leaks are usually seen from 2 to 5 post-operative day presenting as bilious drain from the drain site.

Fistula may be high or low output, in case of high output fistula, where TPN facilities are available, patients can be managed conservatively, and feeding jejunostomy can be done for enteral feeds. Trial can be given for

3 weeks after which operative approach can be adopted.

Where TPN facilities are not available, ideally after resuscitation patient can be taken up for surgery at an early stage to prevent further deterioration. In low output fistula conservative management is usually adopted.

SURGICAL MANAGEMENT OF BILE LEAK:

- Feeding jejunostomy.
- Use of serosal patch (Kobald & Thai) technique: The upper jejunum is used as a loop or Roux-en-y loop to occlude or patch the perforation. Duodenum mucosa lines the serosa.
- Partial gastrectomy: In large perforation with friable margins where repair cannot be done, it is ideal to proceed with partial gastrectomy with Polya anastomosis.
- Conservative or non-operative management: By passing Foley's catheter into the drain wound and manipulating it in to or hear to the perforation can be converted into a controlled fistula.

ANTI ULCER DRUGS

H2 BLOCKER ANTAGONISTS: These drugs act by selectively blocking H2 receptors of parietal cells. They have a dose dependent ant secretory potency. Their simple dosage schedules are associated with good therapeutic compliance.

RAN ITIDINE: l 50mg twice daily, after 4 - 6 wks. once daily at night. FAMOTIDINE: 20 -40 mg OD., NIZATIDINE: 20mg 3D.

PROTON PUMP INHIBITORS: These act by inhibiting the H /K AIR system on the luminal side of the parietal ce Action is long lasting and dose dependent. Omeprazole in dose of 20 -40mg OD achieves almost 100% inhibition of intragastric acidity throughout day and night. Dosage 20-40mg OD for 4 -6 weeks, followed by 10 -20mg OD.

H.PYLORI ERADICATION

The key success factor in management of peptic ulcer is treatment of H• pylon infection, which has been widely advocated current regimen for eradication of H Pylon infection are quite diverse, not only in the combination of agents but also in dosage and duration of the treatment.

There are various regimens against I-I-Pylon;

- Dual drug therapy.
- Triple drug therapy.
- Quadruple drug therapy.

1. DUAL DRUG THERAPY:

- Proton pump inhibitor plus clarithromycin I amoxicillin.
- Ranitidine plus clarithromycin for 14 days. This is not recommended because of its suboptimal results.

2. TRIPLE DRUG THERAPY:

- Omeprazole 40mg OD plus Clarithromycin 500mg BD plus
- Metronidazole 400mg BD for 7days.
- Omeprazole 40mg OD plus Amoxicillin 500mg TID plus
- Clarithromycin 500mg BD for 7 days.
- Omeprazole 40mg 00 p Amoxicillin 500mg TID plus Metronidazole 400mg TID for 7 - 10 days.
- Colloidal Bismuth Sub citrate 125mg OlD plus Amoxicillin 500mg BD plus Metronidazole 400mg TID for 14 days.

3. QUADRUPLE DRUG THERAPY:

- Omeprazole 40mg BD plus Colloidal Bismuth Sub citrate 125mg plus Tetracycline 500mg TID plus Metronidazole 400mg TID for 7 days.
- The Clarithromycin based regimens are much costlier than Amoxicillin based regimens.

NON- OPERATIVE OR CONSERVATIVE MANAGEMENT OF PPU

In majority of patients surgery remains treatment of choice. In certain conditions conservative management should be considered.

Indications for conservative management:

- Where general conditions of the patient is bad, risk of anaesthesia is considered too great.
- Lack of surgical facilities.
- Clinical signs suggesting only of minimal spillage with sealed off perforation which has been shown by gastrograffin radiograph.

Contraindications for conservative management:

- Perforation in presence of steroids, which would diminish the patient's ability to heal the ulcer spontaneously.
- Gastric ulcer.
- Patients who have continued leakage on gastrograffin radiograph.
- Patients who perforate while on active antacid therapy.
- Uncertain diagnosis.

CONSERVATIVE MANAGEMENT CONSISTS OF:

- Nil by mouth regimen.
- Continued nasogastric aspiration.
- Intravenous fluids.
- Intravenous H2 receptor antagonist.
- Appropriate antibiotics.
- Appropriate sedative.

If the distension of abdomen increases with the condition deteriorating, then the flank drain (bilateral) may be placed under local anaesthesia tp drain the fluid.

Advantages of conservative management:

- Operation can be avoided
- A percentage of patients do not need any further definitive operation, in such patients unnecessary operations can be prevented.
- In few patients when perforation will get sealed off and such patients would be benefitted.

Disadvantages of conservative management:

- The site of perforation usually remains in doubt.
- The nature of underlying condition (benign or malignant) remams uncertain.
- Recurrence of ulcer symptoms.
- Recurrence of perforation.
- Risk of deterioration.

PROGNOSIS

Many factors influence the speed at which the peritoneal cnsts develops. Mortality depends upon the following factors:

- Age: mortality increases with increasing age.
- General condition of the patient: poor general condition of the patient carries high mortality.

- Presentation with shock (systolic BP <90 mm Hg): patients presenting with shock will have a high mortality rate.
- Presentation with renal failure: Oliguria carries high mortality which may be due to hypovolemia or septicemia.
- Ulcer history: patients with long history of peptic ulcer disease has shown to carry high risk and concomitant bleeding and perforation also carry high mortality rate.
- Preoperative preparation of the patient also determines the outcome of surgery
- Concomitant medical illness: concomitant cardiac, pulmonary, renal and other disease in association with perforation carry high mortality high.
- Duration of perforation: if the duration is of longer duration, high mortality rate is seen.
- Size of perforation: in duodenal ulcer, the size varies from 3mm to 1 cm in diameter, but in gastric ulcer it may be bigger. Larger the perforation and older the patient, higher the morbidity and mortality.
- Number of perforation: perforation is always single, but there are reports of more than one perforation. More than one perforation, then higher is the morbidity and mortality.

- Nature of gastric contents kind of microorganism which predominantly cultured: in a study conducted by Grarco and Chawo in 1974 and Boey in 1982 cultures of peritoneal fluid taken at the time of operation in early cases were found to be sterile. Bacterial peritonitis is seen in grossly neglected and lately reported cases.

POSTOPERATIVE COMPLICATIONS:

Complications are most likely to happen in higher risk patients. The most common complications in first 24-48 hours after duodenorraphy are

- Inadequate fluid loading and oliguria
- Intraperitoneal abscess, usually sub phrenic or pelvic.
- Wound infection.
- Gastric and duodenal fistulae.
- Respiratory complications: Atelectasis, sometimes preceding pneumonia is common. A persistent basal effusion may require treatment and this complication is an indication of sub diaphragmatic sepsis.

Hennessy in 1969, reporting on 603 cases of perforated gastric and duodenal ulcer found post-operative pneumonia in 42.9% of cases and intra-abdominal abscess in 8.4% and wound infection in 31.9%.

MATERIALS AND METHODS

METHODOLOGY

From August 2016 to July 2017 patients diagnosed with perforative peritonitis with hemodynamic instability in GRH Madurai will be recruited in this study.

A total of 50 patients diagnosed with perforative peritonitis with hemodynamic instability and unfit for immediate laparotomy were selected. Consent obtained from patients and their care-givers. A detailed clinical history regarding age, sex, occupation, clinical presentation, symptoms, past history of chronic duodenal ulcer and history of co-morbidities like diabetes/CAD/hypertension/LRI was obtained. Patients were segregated into control and test groups. Time since onset of pain, pulse rate, BP, respiratory rate, temperature, ABG analysis, IAP, urine output are all noted for test and control groups.

In control group resuscitation is done with IV fluids, inotropes. After stabilization laparotomy and omentalpatch closure done.

In test group, in addition to IV fluids and inotropes, flank drain insertion is done under ultrasound guidance. Quantity and nature of toxic fluid drained is noted. After stabilization laparotomy and omental patch closure is done.

Site and size of perforation, type of anesthesia, intra-op hypotension and arrhythmia, post-op mechanical ventilation, inotrope requirements, atelectasis, urine output, IAP, return of bowel sounds are all noted for test and control groups

All patients were started on IV antibiotics for a minimum of 5 days. All patients were placed on injectable analgesics, tapered as they recovered. All patients were starts on proton pump inhibitors. Nasogastric tube was placed, continuous drainage was done till the secretions were less than 150 ml in 24 hrs. Patients were allowed to take liquid diet once the bowel sounds returned. Solid diet was allowed as the patient tolerated. Abdominal drains were monitored and removed when collection was less than 50 ml in 24 hrs. Patients were encouraged to move about, as their condition allowed. Patients were monitored for fever, post operative complications, respiratory infection. Patients were discharged once they were free of post-operative complications, able to move about comfortably and the sutures healed. Patients were prescribed 3 drug anti-H.pylori regimen at the discharge and were advised to come for follow up at 2 weeks, 1 month and 3 months from discharge.

ELIGIBILITY CRITERIA

A. Inclusion criteria:

1. Patients of age 25-60 years diagnosed with perforative peritonitis with hemodynamic instability (BP</= 100/60) in GRH Madurai.
2. Patients consented for inclusion in the study according to designated proforma

B. Exclusion criteria:

1. Patients who have loculated fluid collections in peritoneum as detected by ultrasound
2. Patients who are hemodynamically stable(BP> 100/60) to undergo immediate laparotomy
3. Patient not consented for inclusion in the study.

STATISTICAL METHODS

Statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (M and results on categorical measurements are presented in Number(%).Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups. Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups. P-value 0.05 is considered as significant.

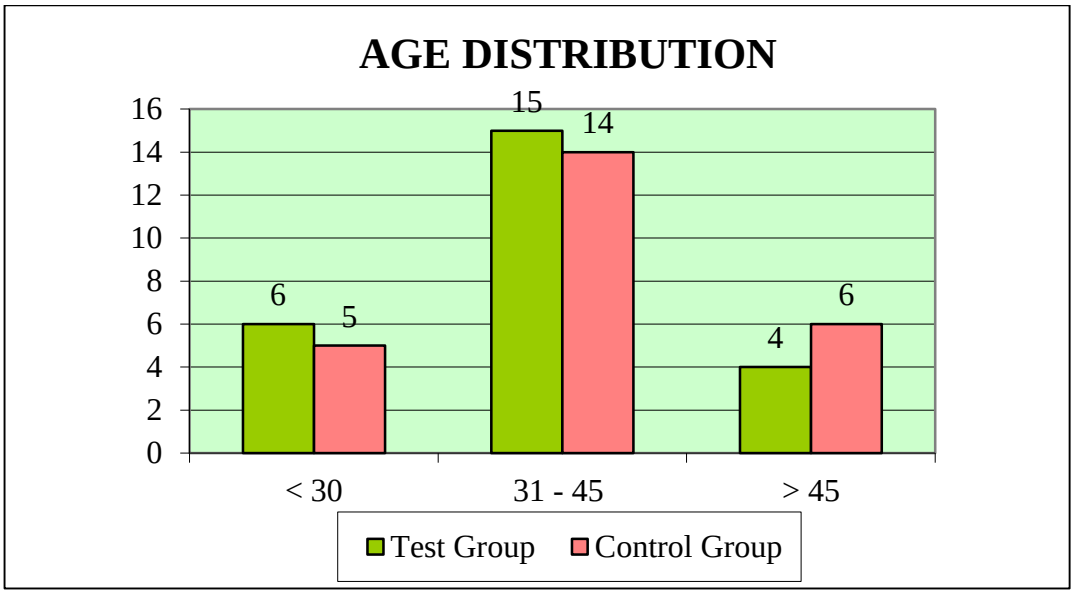
Statistical software: The Statistical software namely SPSS 150, Stats Direct and Systat11.0 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs and tables.

RESULTS AND INTERPRETATION

OBSERVATION AND RESULTS

AGE DISTRIBUTION

Age in years	Test Group	Control Group
< 30	6	5
31 - 45	15	14
> 45	4	6
Total	25	25
Mean	37.08	38.08
SD	9.3	10.53
p value	0.723 Not significant	

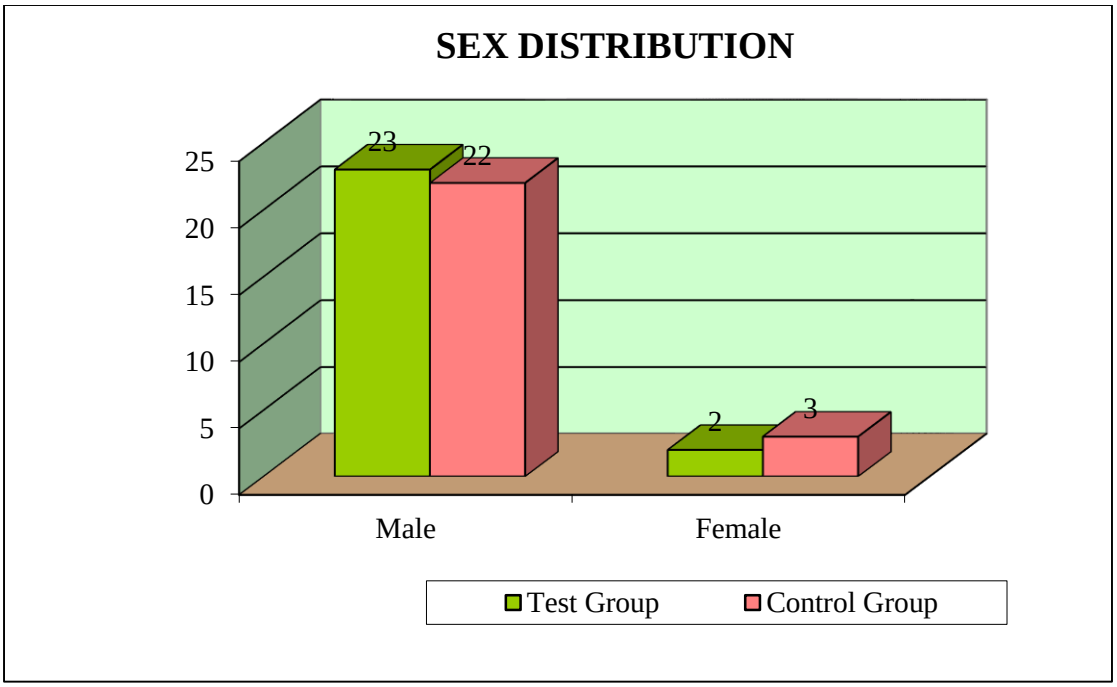


Patients were randomly assigned to the test and control groups. The mean age test and control groups were 37.08 and 38.08 respectively. The p value is 0.723 thus making it not significant.

Hence the distribution of cases according to age does not influence the results of the study.

SEX DISTRIBUTION:

Sex	Test Group	Control Group
Male	23	22
Female	2	3
Total	25	25
pvalue	0.865 Not significant	

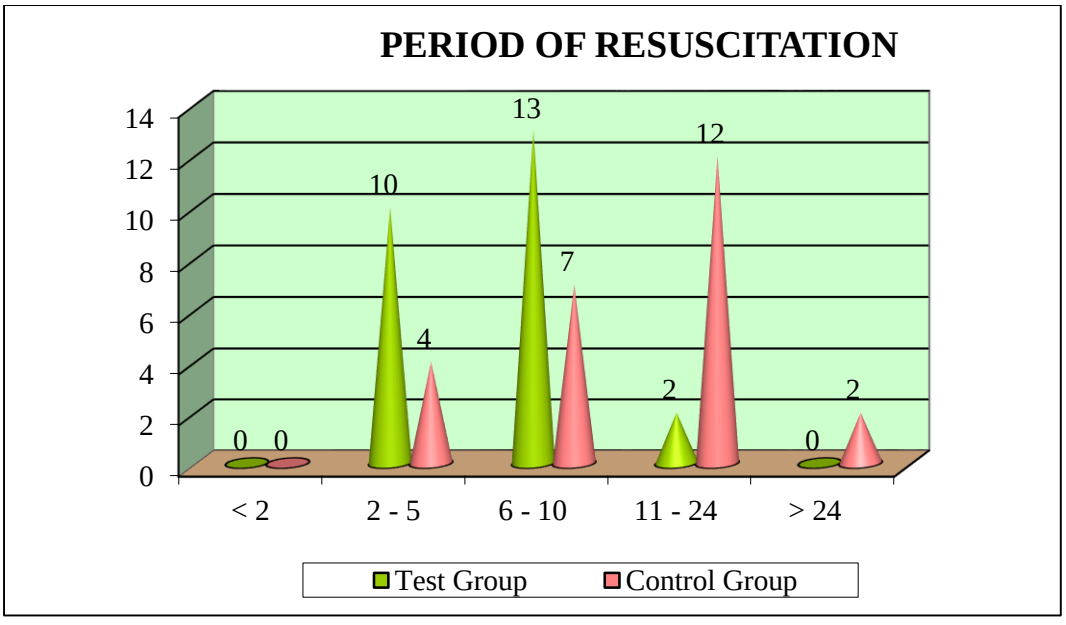


Most of the cases of perforative peritonitis encountered were males.In the study conducted, 23 patients in the test group and 22 cases in the control group were males and 2 cases in the study group and 3 cases in the control group were females. The p value of sex distribution is 0.865.Thus the distribution of sex between the groups is comparable.

PERIOD OF RESUSCITATION:

Period of Resuscitation (Hrs)	Test Group	Control Group
< 2	0	0
2 - 5	10	4
6 - 10	13	7
11 - 24	2	12
> 24	0	2
Total	25	25

Period of Resuscitation (Hrs)	Test Group	Control Group
< 10	23	11
> 10	2	14
Total	25	25
p value	< 0.001 significant	



23 out of 25 patients in test group were resuscitated within 10 hours of admission whereas only 11 out of 25 patients in control group could be resuscitated within 10 hours. The p value is <0.001 which is significant. This shows that insertion of flank drain significantly reduces the period of resuscitation.

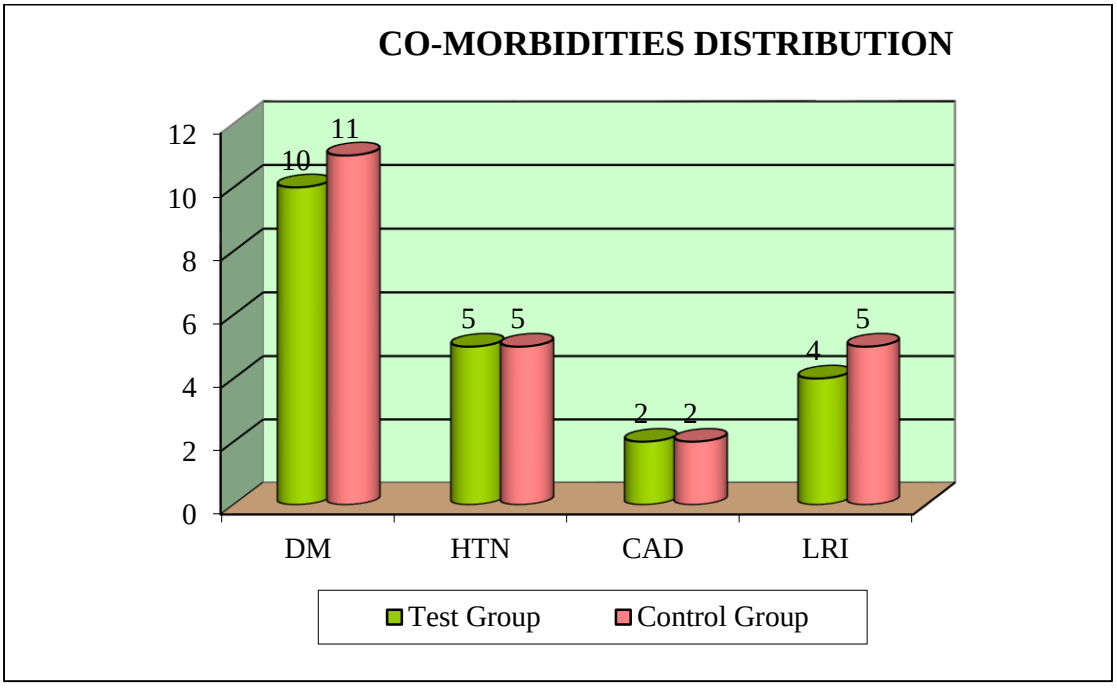
TIME SINCE PAIN ONSET:

Time since pain onset (Hrs)	**Test Group**	**Control Group**
Mean	17.24	18.08
SD	12.57	13.75
p value	0.823 Not significant	

Time since pain onset is the time between onset of pain and admission. The p value is 0.823(not significant). Since it can act as confounding factor, time since pain onset is equally distributed between test and control groups.

CO-MORBIDITIES:

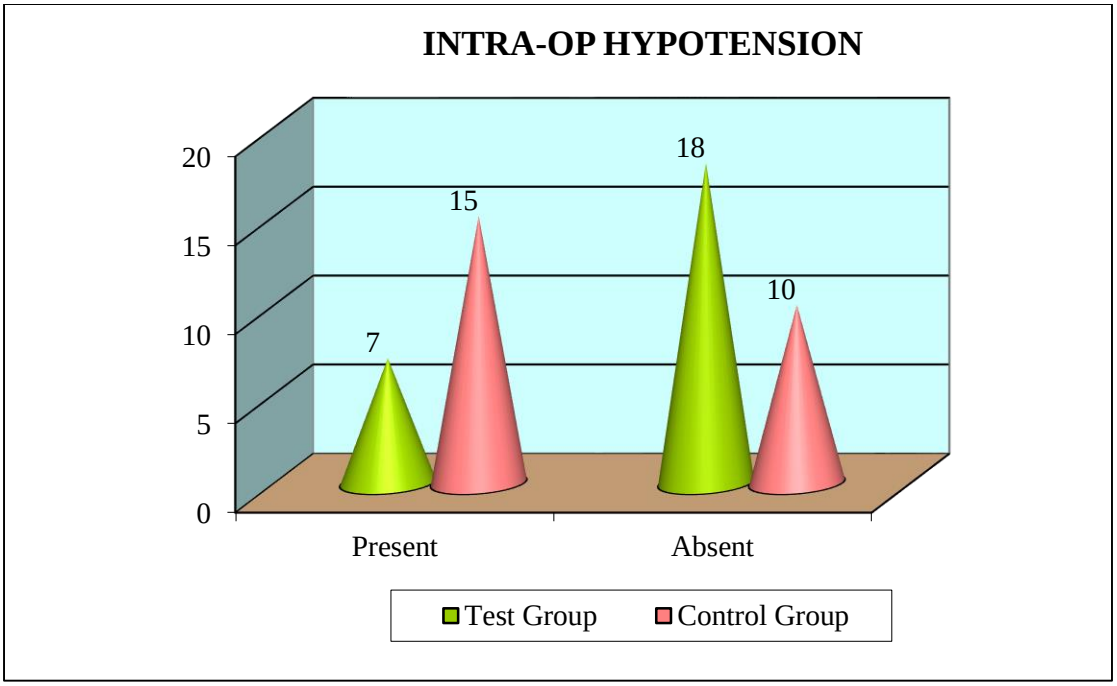
Co-morbidities	Test Group	Control Group
DM	10	11
HTN	5	5
CAD	2	2
LRI	4	5



The distribution of co-morbidities like diabetes mellitus, hypertension, coronary artery disease and lower respiratory tract infections among test and control groups were randomized so that it does not influence the study results.

INTRA-OP HYPOTENSION:

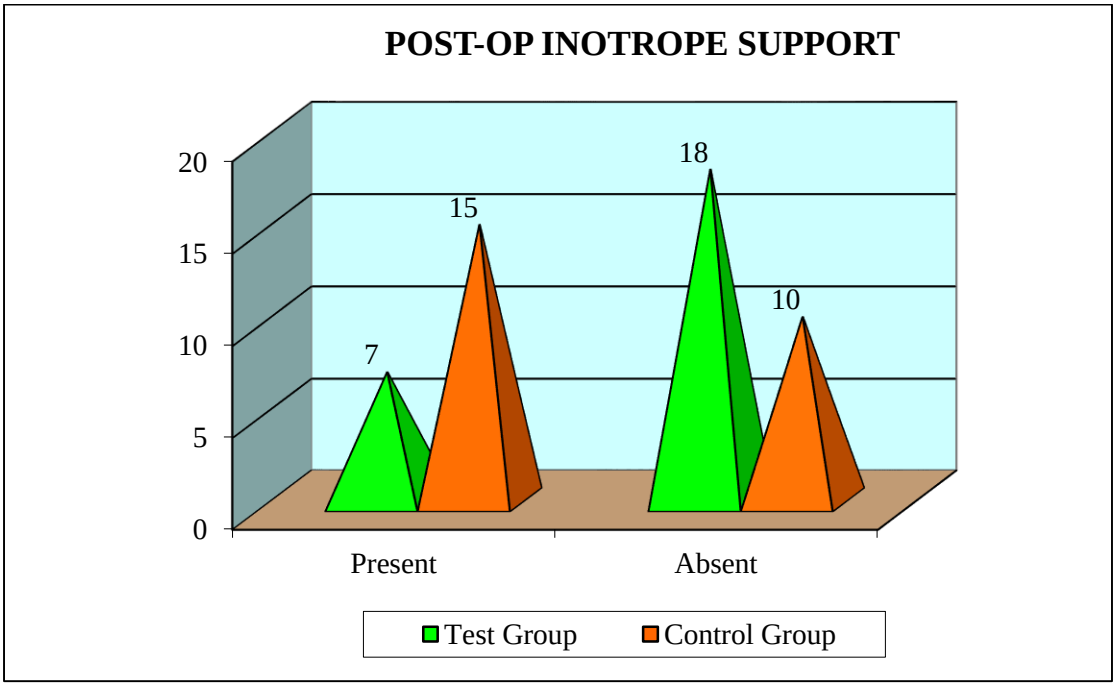
Intra-op Hypotension	Test Group	Control Group
Present	7	15
Absent	18	10
Total	25	25
p value	0.046 Significant	



Intra-op hypotension occurred in 7 out of 25 patients in test group and 15 out of 25 patients in control group. The p value is 0.046(significant). Hence insertion of flank drain during resuscitation significantly decreases the incidence of intra-op hypotension.

POST-OP INOTROPE SUPPORT:

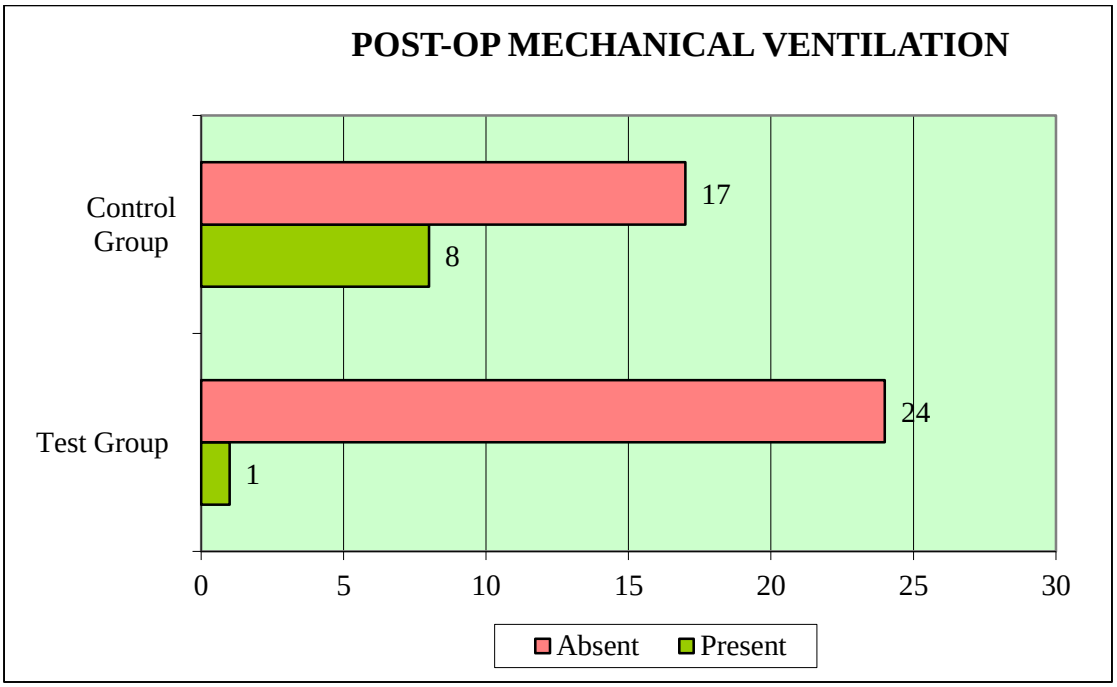
Post-op inotrope support	Test Group	Control Group
Present	7	15
Absent	18	10
Total	25	25
p value	0.046 Significant	



All patients who developed intra-op hypotension required inotropic support post-operatively. Inotropic support was required in in 7 out of 25 patients in test group and 15 out of 25 patients in control group. The p value is 0.046(significant).Hence insertion of flank drain during resuscitation significantly decreases the requirement of post-op inotropic support.

POST-OP MECHANICAL VENTILATION:

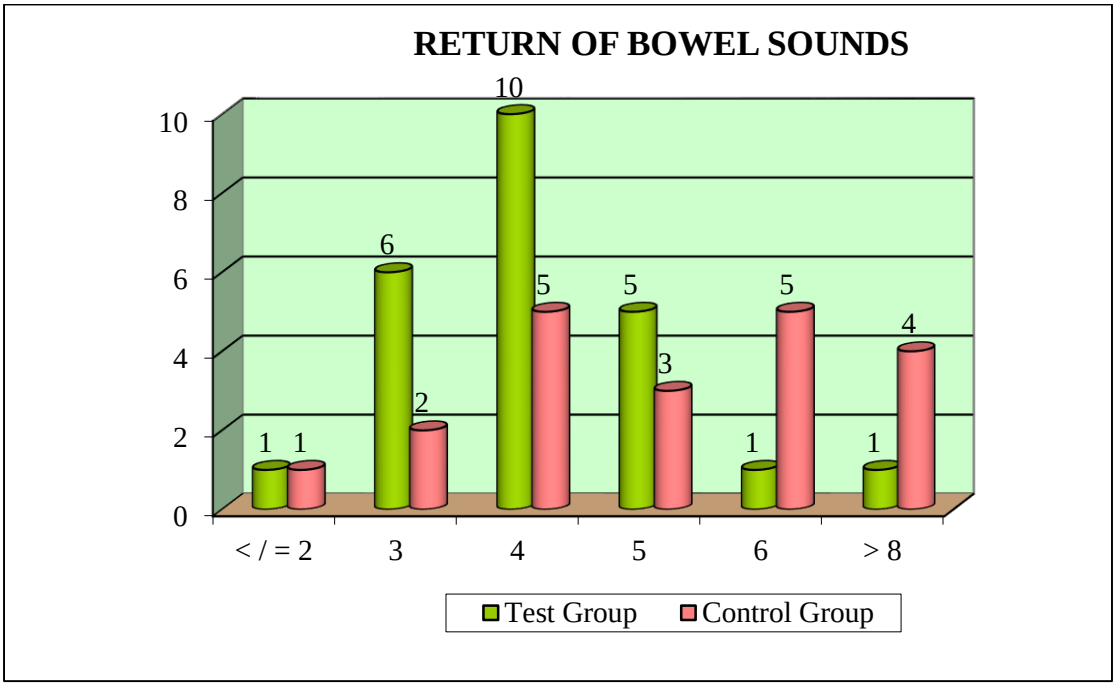
Post-op Mechanical ventilation	Test Group	Control Group
Present	1	8
Absent	24	17
Total	25	25
p value	0.023 Significant	



Post-op mechanical ventilation was required in in 1 out of 25 patients in test group and 8 out of 25 patients in control group. The p value is 0.023(significant). Thus insertion of flank drain during resuscitation significantly decreases the requirement of post-op ventilatory support and hence decreases the morbidity of the patients.

RETURN OF BOWEL SOUNDS:

Return of Bowel Sounds (Days)	Test Group	Control Group
< / = 2	1	1
3	6	2
4	10	5
5	5	3
6	1	5
> 8	1	4
Total	25	25

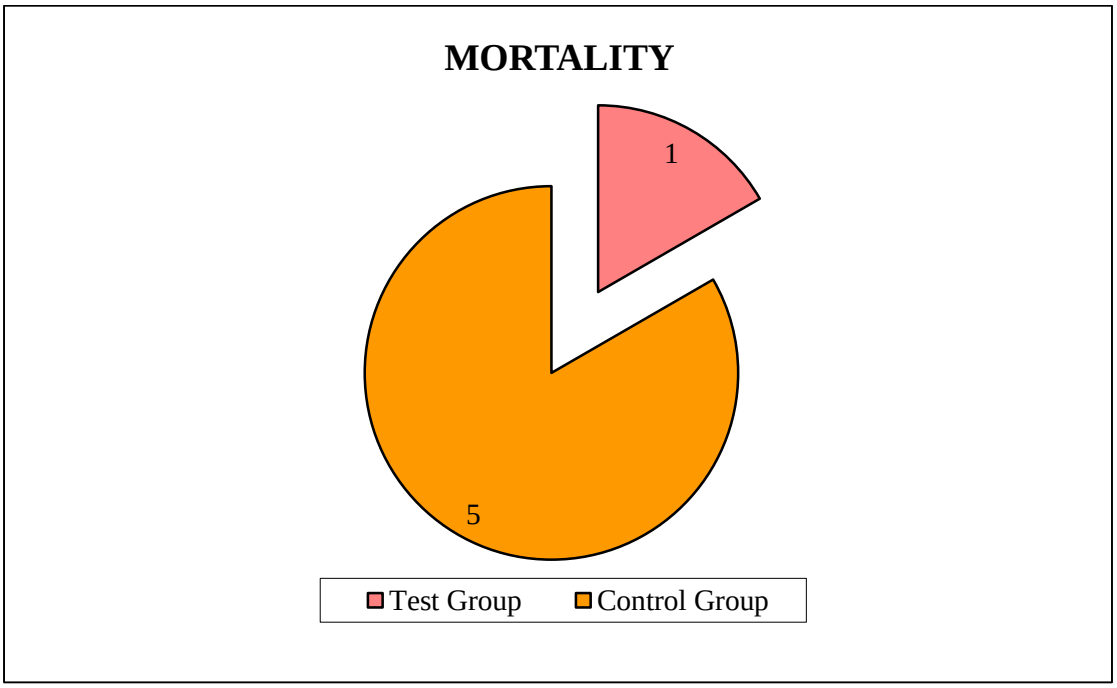


Return bowel sounds	Test Group	Control Group
< 5 days	22	11
> 5 days	2	9
Total	25	25
	p value	0.041 significant

Bowel sounds resumed within 5 days postoperatively in 22 out of 25 patients in test group and 11 out of 25 patients in control group. The p value is 0.041. This shows that the patients in test group had fastened recovery and hence less morbidity.

MORTALITY:

Outcome	Test Group	Control Group
Mortality	1	5
p value	0.189 Not significant	



1 out of 25 patients in test group and 5 out of 25 patients in control group expired. Hence insertion of flank drain during resuscitation reduces mortality and improves outcome of patients.

DISCUSSION

DISCUSSION

The indications for surgery in PUD are bleeding, perforation, obstruction, and intractability or nonhealing. Perforation is the second most common complication of peptic ulcer but nowadays a more common indication for operation than bleeding. This study is concerned with the comparison of the perioperative outcomes of cases of perforative peritonitis undergoing/not-undergoing flank drain insertion during resuscitation prior to laparotomy and the scope to implement the same.

For analysis of the patient outcome, the following parameters were taken into account:

1. Period of resuscitation
2. Intra-op hypotension
3. Post-op inotropic support
4. Post-op ventilatory support
5. Return of bowel sounds

- Period of resuscitation is significantly lower in test group.
- Occurance of intra-op hypotension and requirement of post-op inotropic support were high in control group compared to test group

- Requirement of post-op ventilatory support is high among control group than test group
- Return of bowel sounds were earlier among test group than controls.
- Mortality was high among controls than test group.

Based on the above analysis, it is evident that in cases of perforative peritonitis unfit for immediate laparotomy, insertion of flank drain during resuscitation can decrease the morbidity and mortality of the patients and improve their perioperative outcomes.

In a study by Baloch .et.al which included 50 patients with perforative peritonitis who are unstable and not fit for immediate laparotomy, flank drain insertion was done which improved the outcome. However there was no control group to compare the results which is overcome in the present study.

CONCLUSION

CONCLUSION

Management of sepsis is a challenge in treating patients with perforation peritonitis.

Percutaneous placement of flank drains under ultrasound guidance during resuscitation prior to laparotomy may act as a temporizing measure by reducing intra- abdominal pressure of patients with perforation peritonitis. It can lead to improvements in their physiological status and significantly contributes to their resuscitation and postoperative outcome.

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PROFORMA

PROFORMA

Name: Unit:

Age: D.O.A:

Sex: D.O.D.:

Diagnosis :

Alcohol abuse :

Analgesic abuse :

Time since on set of pain :

Diabetes :

Hypertension :

CAD :

LRI :

Other co-morbidities :

Pre-Op Parameters

	Pre Insertion	**Post Insertion**		
		1 Hr	**2 Hrs.**	**3 Hrs**
Pulse Rate				
Blood Pressure				
Respiratory Rate				
Temperature				
Arterial Blood Gas				
Intra Abdominal Pressure				
Urine Output				

Quantity of Toxic fluid drained :

Nature of fluid drained :

Period of Resuscitation :

Intra-Op Parameters

Site of Perforation :

Size of perforation :

Type of Anaesthesia :

Intra-Op Hypotension :

Intra-Op Arrhythmia :

Post-Op Parameters

Inotrope Support :

Mechanical Ventilation :

Atelectasis :

Wound site infection :

	Day 1	**Day 2**	**Day 3**	**Day 4**	**Day 5**
Bowel sounds					
Passing flatus					
Ryle's tube aspirate					
Urine output					
Intra abdominal pressure					

ABBREVIATIONS

ABBREVIATIONS

DM	-	Diabetes Mellitus
HTN	-	Hypertension
CAD	-	Coronary Artery Disease
LRI	-	Lower Respiratory tract Infection
M	-	Male
F	-	Female
P	-	Present

MASTER CHART

MASTER CHART – CONTROL GROUP

S.NO.	NAME	AGE	SEX	PERIOD OF RESUSCITATION (HRS)	TIME SINCE PAIN ONSET	CO-MORBIDITIES				INTRA - OP HYPO TENSION	POST-OP IONO TROPE SUPPORT	POST-OP MECHANICAL VENTILATION	RETURN OF BOWEL SOUNDS (DAYS)	MORTALITY
						DM	HTN	CAD	LRI					
1	RAJENDRAN	60	M	3	11	P	P	P	P	P		P		DEATH
2	MUTHU IRULANDI	25	M	12	10								2	
3	LAKSHMI	32	F	8	9					P			5	
4	VEERABADRAN	37	M	4	48								3	
5	SARAVANA KUMAR	55	M	14	22	P			P	P		P		DEATH
6	MUTHUKRISHNAN	30	M	12	6								4	
7	SETHUPATHY	35	M	6	31	p				P			6	
8	CHANDRAN	35	M	28	22								8	
9	PRABHU	39	M	4	13	P				P		P	6	
10	MANIMARAN	58	M	18	12	P	P		P	P		P		DEATH
11	RAMAR	25	M	12	11								3	
12	AROKYASAMY	38	M	15	15	P				P			6	
13	Mary	34	F	10	35								5	
14	GOPI	32	M	2	8								8	
15	RAMASAMY	48	M	16	11	P	P	p		P		P		DEATH
16	RAJAMANI	33	M	24	48								6	
17	CHINNAYA	38	M	9	7	P				P			6	
18	KARUPASAMY	47	M	30	52					P			5	
19	MUNIYANDI	31	M	7	11								3	
20	LINGAMMAL	56	F	20	6	P	P		P	P		P		DEATH
21	RAJA	35	M	10	13				p				4	
22	POOMURUGAN	40	M	14	15	P				P		P	8	
23	VINEETH	40	M	16	8	P	P			P		P	8	
24	PALANISAMY	28	M	20	12					P			5	
25	KALIMUTHU	25	M	8	16								2	

MASTER CHART – TEST GROUP

S. NO.	NAME	AGE	SEX	PERIOD OF RESUSCITATION (HRS)	TIME SINCE PAIN ONSET (HRS)	CO-MORBIDITIES				INTRA-OP HYPOTENSION	POST-OP IONO TROPE SUPPORT	POST-OP MECHANICAL VENTILATION	RETURN OF BOWEL SOUNDS (DAYS)	MORTALITY
						DM	ATN	CAD	LRI					
1	PARTHIBAN	25	M	2	7								2	
2	RAJARAM	30	M	4	20								4	
3	VALLI	35	M	5	12								5	
4	SANKARAN	36	M	6	16	P	P				P		5	
5	MUTHUKUMAR	44	M	9	35	P				P	P		4	
6	AZHAGAR	32	M	8	42								4	
7	AMMASI	29	M	3	5								2	
8	RAKESH	36	M	7	53								3	
9	MAHARAJAN	38	M	8	7	P							4	
10	SELVI	55	F	10	11	P	P		P	P		P	8	
11	ESAKIPANDI	56	M	2	10	P	P	P	P	P	P			DEATH
12	GANESHAN	40	M	7	9	P				P			5	
13	KESAVAN	39	M	4	8								3	
14	RAMAYEE	31	F	9	22						P		3	
15	DHARMARAJ	48	M	5	6	P	P		P	P			5	
16	DHIVAKAR	33	M	7	31								4	
17	MUNIASAMY	28	M	6	22								3	
18	MOHAMMAD FARAZ	34	M	7	13						P		4	
19	GUNASEELAN	36	M	3	12								5	
20	PITCHAI	58	M	9	11	P	P	P	P	P			6	
21	MOORTHY	25	M	3	15						P		5	
22	RAMAIAH	37	M	12	35								3	
23	GAJENDRAN	38	M	5	8	P				P	P		4	
24	SHANKAR	27	M	15	11								3	
25	SONGU	40	M	10	10	P				P	P	P	4	

ETHICAL COMMITTEE APPROVAL LETTER

ETHICAL COMMITTEE APPROVAL LETTER

MADURAI MEDICAL COLLEGE
MADURAI, TAMILNADU, INDIA -625 020
(Affiliated to The Tamilnadu Dr.MGR Medical University, Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.Thalabathik kumara vikram S
Course	:	PG in MS., General Surgery
Period of Study	:	2015-2018
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Prospective study on impact of USG guided flank drain insertion on the perioperative outcome of cases of perforative peritonitis with hemodynamic instability in GRH, Madurai
Ethical Committee as on	:	02.06.2017

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary

Chairman
Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
Madurai

Dean/Convenor
Madurai Medical College
Madurai-20

ANTI PLAGIARISM CERTIFICATE

ANTI PLAGIARISM CERTIFICATE

URKUND

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